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EXAMINER
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ART UNIT PAPER NUMBER

1647

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Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary

Application No.
09/292,862

Applicant(s)
Walter et al

Examiner
Sharon L. Turner, Ph.D.

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-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136 (a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 1-11-02
- 2a) ☒ This action is FINAL. 2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 35 C.D. 11; 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 1-17 is/are pending in the application.
- 4a) Of the above, claim(s) 6-7, 9-10, 12-14 and 15-17 to the extent of claim 7 is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 1-5, 8, 11, and 15-17 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☒ Claims 1-17 are subject to restriction and/or election requirements.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are objected to by the Examiner.
- 11) ☐ The proposed drawing correction filed on _____ is: a) ☐ approved b) ☐ disapproved.
- 12) ☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. § 119

- 13) ☐ Acknowledgement is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d).
- a) ☐ All b) ☐ Some* c) ☐ None of:
- ☐ Certified copies of the priority documents have been received.
 - ☐ Certified copies of the priority documents have been received in Application No. _____.
 - ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- *See the attached detailed Office action for a list of the certified copies not received.
- 14) ☐ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. § 119(e).

Attachment(s)

- 15) ☒ Notice of References Cited (PTO-892)
- 16) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 17) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s). _____
- 18) ☐ Interview Summary (PTO-413) Paper No(s). _____
- 19) ☐ Notice of Informal Patent Application (PTO-152)
- 20) ☐ Other: _____

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Response to Amendment

1. The amendment filed 1-11-02 has been entered into the record and has been fully considered.
2. The text of Title 35 of the U.S. Code not reiterated herein can be found in the previous office action.
3. As a result of applicants amendment, all rejections not reiterated herein have been withdrawn by the examiner.

Election/Restriction

4. Claims 1-17 are pending. Applicants election of Group I, claims 1-5, 8, 11 and 15-17 without traverse in Paper No. 11 is acknowledged
5. Claims 6-7, 9-10, 12-14 and 15-17 to the extent of claim 7 are withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to a nonelected invention, there being no allowable generic or linking claim. Election was made **without** traverse in Paper No. 11.

Sequence Compliance

6. Figure 2 lacks full sequence compliance as the Brief Description references only the nucleic acids denoted in the figure and identified as SEQ ID NO:1. However, it is noted that the figure also discloses an amino acid sequence which is not referenced by an appropriate sequence identifier. Correction is required.

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Rejections Maintained

Claim Rejections - 35 USC § 112

7. The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

8. Claims 1-5, 8, 11 and 15-17 stand rejected under 35 U.S.C. 112, first paragraph, as set forth in Paper No. 15, mailed 6-28-01 as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. This is a written description rejection.

The specification discloses SEQ ID NO's: 1 and 2 which correspond respectively to the nucleotide sequence and the amino acid sequence of the human FREAC3 gene. These SEQ ID NO's meet the written description provisions of 35 USC 112, first paragraph. However, the claims are directed to and encompass genes, corresponding sequences from other species, mutated sequences, allelic variants, and splice variants. None of these sequences meets the written description provision of 35 USC 112, first paragraph. In particular it is noted that as recognized by Lewin Ed., Genes IV, Oxford University Press 1990, p. 810, a gene encompasses that segment of DNA involved in producing polypeptide chains including regions preceding and following the coding region (leader and trailer) as well as intervening sequences (introns) between coding regions (exons). Although SEQ ID NO:1 contains some sequences upstream and

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downstream such sequences are not recognized or disclosed as the full leading and trailing sequences which are responsible for the regulated gene expression of the polypeptide via mRNA within the natural host, i.e., the full promoter and trailer sequences of the DNA from which the mRNA is transcribed.

Vas-Cath Inc. v. Mahurkar, 19 USPQ2d 1111, makes clear that, "applicant must convey with reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession of the invention. The invention is for purposes of the 'written description' inquiry, whatever is now claimed." (See Vas-Cath at page 1116.)

With the exception of SEQ ID NO's:1 and 2 of the instant application, the skilled artisan cannot envision the detailed chemical structure of the encompassed nucleic and amino acid sequences and therefore conception is not achieved until reduction to practice has occurred, regardless of the complexity or simplicity of the method of isolation. Adequate written description requires more than a mere statement that it is part of the invention and reference to a potential method for isolating it. The specific nucleic and amino acids are required. See Fiers v. Revel, 25 USPQ2d 1601, 1606 (CAFC 1993) and Amgen Inc. v. Chugai Pharmaceutical Co. Ltd., 18 USPQ2d 1016.

One cannot describe what one has not conceived. See Fiddes v. Baird, 30 USPQ2d 1481, 1483. In Fiddes v. Baird, claims directed to mammalian FGF's were found unpatentable due to lack of written description for the broad class. The specification provided only the bovine sequence.

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Therefore, only SEQ ID NO's:1 and 2, but not the full breadth of claims meet the written description provision of 35 USC 112, first paragraph. Applicant is reminded that Vas-Cath makes clear that the written description provision of 35 USC 112 is severable from its enablement provision. (See page 1115.)

Applicants argue that claim 1 has been amended to require that the FREAC3 gene encode a polypeptide of 90% identity to human FREAC3 and argue that the nucleic acids were envisioned and reduced to practice at the time of filing. Applicants argue that p. 14, lines 15-23 provide support for FREAC3 genes which are substantially identical to human and that specific mutations, truncations and mammalian homologues are described. Further notations of these variants and of murine FREAC3 (Mfl protein) sharing 89% homology are further supported at p. 10, lines 12-14, page 40, line 20 and p. 42 through p. 44. Applicants also argue that they have disclosed at least three functional mutations and/or allelic variants of FREAC3 gene and that thus the invention is adequately described and the rejection should be withdrawn.

Applicant's arguments filed 1-11-02 has been fully considered but are not persuasive. The examiner notes as previously set forth that the recitation of "a gene" requires a written description for more than the mere coding sequence. Upstream and downstream regulatory sequences are included in the recitation but are not described. In addition, while applicants appear to have described a few members of FREAC3 proteins, including members from different species and mutated or allelic variants of FREAC3 proteins, the description of the members is inadequate to describe the extent to which each genus exhibits variability and the extent to which

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the two genus' are separate, as encompassed by the claims. For example, what guidance is the artisan to use to determine which of the described 90% identical sequences correspond to FREAC3 genes and which of the described 90% identical sequences correspond to FREAC3 mutations? The description approximates FREAC3 as a general class but does not delineate the extent to which the genus recitations encompass normal or mutated sequences. The differences in sequence which correspond to a FREAC3 gene or protein and those differences which alternatively describe mutated or mutations are not delineated or described as generically recited and thus the claims lack adequate written description of the recited genus' of molecules encompassed.

9. Claims 1-5, 8, 11 and 15-17 stand rejected as set forth in Paper No. 15, mailed 6-28-01, under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for the sequences of SEQ ID NO:1 and 2 which describe a FREAC3 gene product, does not reasonably provide enablement for a method of diagnosing a mammal for an increased likelihood of having a developmental defect or developing a disease of the eye by analyzing nucleic acid of said mammal to determine whether said nucleic acid contains a mutation in a FREAC3 gene wherein the presence of said mutation is an indication of increased likelihood of developing a disease of the eye. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

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The specifications disclosure is insufficient to enable one skilled in the art to practice the invention as broadly claimed without undue experimentation. The factors relevant to this discussion include the quantity of experimentation necessary, the lack of working examples, the unpredictability of the art, the lack of sufficient guidance in the specification and the breadth of the claims.

Applicants claims are directed to the generic recitation of analyzing nucleic acid from a mammal. The skilled artisan recognizes a multitude of experimental techniques sufficient to analyze nucleic acid sequences including for example hybridization, Polymerase Chain Reaction (PCR), Restriction Fragment Length Polymorphism analysis (RFLP), nucleic and amino acid sequencing, homologous recombination, expression studies, in situ Hybridization and Reverse Transcriptase-PCR. However, the skilled artisan also recognizes that the success of such methodologies is unpredictably dependent upon variables such as the nucleic acids employed in the techniques, hybridization conditions, the relatedness of gene sequences, species variation and the inability to predict structural and functional determinants of nucleic and amino acid sequences, see in particular Skolnick et al., Trends in Biotech, 18(1):34-39, 2000 and Sambrook et al., Molecular Cloning, Cold Spring Harbor Laboratory Press, 1989, p9.47-9.51 and 11.48-11.49.

With respect to applicants claims, the skilled artisan is not apprised of that which is identified as a FREAC3 gene because the relevant nucleic and amino acids are absent from the claims. There is no guidance as to what methodologies should be employed to analyze the

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nucleic acids and there is no defined sequences which are identified as either FREAC3, mutated FREAC3 which indicates a developmental defect or disease of the eye, or disclosure of sequences which may be used to analyze for the presence or absence of FREAC3 and FREAC3 mutations. Thus, the skilled artisan would be forced into further undue experimentation to discover the FREAC3 gene sequences, gene products and methodologies sufficient to identify normal and mutated sequences. Further, the skilled artisan would be required to determine which mutations led to either developmental defects or eye disease.

Thus, in view of the quantity of experimentation necessary, the lack of working examples, the unpredictability of the art, the lack of sufficient guidance in the specification and the breadth of the claims, it would take undue experimentation to make and use the claimed invention.

Applicants argue that Sambrook evidences a high skill in the art, that prediction of function solely on structural requirements is not necessary, that the determination of undue experimentation must be evaluated with respect to the guidance provided in the specification, that the artisan is equipped to analyze the nucleic acids and to assess functionality of FREAC3 mutations, for example in silent mutations, anterior segment dysgenesis, and in FREAC3 binding.

Applicant's arguments filed 1-11-02 have been fully considered but are not persuasive. While the skill in the art of nucleic acid analysis is high, the specification fails to teach or delineate the structural and/or functional requirements of a FREAC3 gene from a FREAC3 mutation. As Skolnick evidences, even amongst highly similar families only testing is sufficient

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to confirm the functionality of even highly related sequences because mutations are unpredictable in nature and in the art. The specification fails to teach which changes in nucleic/amino acid structure more likely than not lead to a normal FREAC3 gene, normal binding activity and normal development of the eye, from those changes which lead to a mutated FREAC3 gene, abnormal binding and abnormal eye development leading to anterior segment dysgenesis. It is especially noted that a sequence which is normal for one species of animal may not be normal for another. Additionally any particular species may contain hundreds of normal allelic variant which are not "defective." Yet there is no guidance in the specification with which to reliably gauge the classification a particular sequence may take. Thus, the artisan is required to perform both the structural and the functional analysis in every case, because the classification of any FREAC sequence is unpredictable. Thus, the guidance provided by the specification is insufficient to enable the broad class of molecules and functions claimed because the guidance is insufficient to describe the respective genus' each from the other. At most the specification provides a research plan to discover those sequences which are aberrant from those which are considered naturally occurring allelic variants and which function in line with normal FREAC3. It is further noted that any particular protein may be multi-functional in nature and there is no evidence that the only deficiency occurring due to FREAC3 mutation is abnormal binding and/or development of anterior segment dysgenesis. It would be likely that other abnormalities may occur, yet such mutations and dysfunctions are not described but appear to be encompassed by the scope of the claims which encompasses any mutation and any "developmental defect" or

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"disease of the eye" as recited. It is additionally noted that the claim recites "a likelihood of developing a disease" yet there is no method to assay for the "likelihood" but only a transgenic which exhibits disease. Thus, the amount of experimentation required is necessarily extensive and undue because the significance of each variation not specifically disclosed is unknown to the artisan prior to biological research testing which is designed to discover the molecules real/true function.

10. The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

11. Claims 1-5, 8, 11 and 15-17 stand rejected under 35 U.S.C. 112, second paragraph, as set forth in Paper No. 15, mailed 6-28-01 as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

The recitations of a FREAC3 gene and mutations in a FREAC3 gene are indefinite terms to the skilled artisan as the claims do not structurally define the products such that the skilled artisan can recognize, test for, make or use the desired sequences. In the absence of a structural definition of FREAC3 or a FREAC3 mutation any genetic nucleic acid would meet the limitations of the claims.

Applicants argue that as amended the FREAC3 gene is 90% identical to SEQ ID NO:2, that as claimed there is structural and functional definition to the products and that these criteria clearly define the metes and bounds of the claim.

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Applicant's arguments filed 1-11-02 have been fully considered but are not persuasive. Applicants appear to have provided structural constraints to "said FREAC3 gene" yet the only FREAC3 with antecedent basis in the claim is "a mutation in a FREAC3 gene". Thus the sequences of both the normal FREAC3 gene and the mutated FREAC3 gene are encompassed by 90% identity, although as the "said mutation" is not limited to any particular mutation, the FREAC3 mutated sequences have unlimited structural constraints. As the nucleic acids are not distinguished from each other, they do not result in the recited outcome of distinguishing by nucleic acid analysis whether or not there is a mutation, except for perhaps in the instance that the variability is greater than 90%. Yet for those sequences in which the FREAC3 gene and the FREAC3 mutations share 90% identity to SEQ ID NO:2, there is no determination as recited in the claim (as to whether or not a mutation is present). Thus, the method is incomplete as apparent steps in the "analysis" which result in the capability to distinguish the FREAC3 sequences from normal and mutated, are not present. These steps are critical if not essential to the outcome of the method yet are not included. It is noted that in contrast to applicants assertion there is no functional assessment in the method. The analysis of the nucleic acid as claimed is what is supposed to provide for whether or not "said mammal has an increased likelihood of developing a disease of the eye," not further functional testing to assess such.

Claim Rejections - 35 USC § 102 or 103

12. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

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A person shall be entitled to a patent unless --

(a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

13. Claims 1, 8, 11, 15 and 17 stand rejected under 35 U.S.C. 102(b) as set forth in Paper No. 15, mailed 6-28-01 as being anticipated by Mears et al., Am. J. Hum. Genet., 59:1321-27, 1996.

Mears et al., teach autosomal dominant genetic mutations which cause anterior segment dysgenesis. The mutation maps to chromosomal location 6p25. The disease marks with deletions in humans and results in juvenile glaucoma, see in particular Abstract and Linkage Analysis, p. 1321-22. Although the reference fails to disclose a FREAC3 gene or mutation the nucleic acids analyzed at 6p25 meet the limitations of a genetic nucleic acid sequence which may be recognized or named such by the skilled artisan absent factual evidence to the contrary or defining structural limitations of FREAC3. Thus, the reference teachings anticipate the claimed invention.

Applicant's argue that Mears is not enabling for the IGDA mutation noted to cause anterior segment dysgenesis because they only isolated the mutation to within 8.3 cM of nucleic acid sequence and that this sequence represents multiple genes. Applicants further argue that no specific mutation is defined and that in contrast, the instant invention is a sequence which encodes a polypeptide within 1662 nucleotides with identified structural and functional domains as well as allelic variants and inactivating mutations. Applicants argue that Watson note that cloning a gene 5cM away from a genetic marker is not trivial.

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Applicant's arguments filed 1-11-02 have been fully considered but are not persuasive. In particular it is pointed out that as applicants argue and the examiner agrees (see enablement rejection above), the skill in the art of nucleic acid analysis is high. Mears evidences isolation of two separate nucleic acid sequences located at the 6p25 segment, one which possess a mutation that causes anterior segment dysgenesis and one which is normal, and/or not mutated in a way to cause such disease. It is further noted that the breadth of the claims includes any degree of variation for the mutated sequence and that the nucleic acid comparison required to determine the actual sequences of the 8.3 cM segment involves sequencing techniques such as "walking" which are routine in the art, see in particular Kleff et al., EMBO J., 11(2):699-704, 1992 and Shirley et al., Plant Cell 4(3):333-47, 1992, and is capable of defining the specific differences amongst the isolated sequences. In addition, it is noted that the sequence characteristics of the claims are inherently provided as the FREAC3 gene contained within the chromosomal region is deemed to be at least 90% identical to SEQ ID NO:2, absent convincing factual evidence to the contrary. It is noted that the Kleff and Shirley references provide support as to the skill in art of chromosomal walking and nucleic acid sequence analysis. Thus, the reference teachings anticipate the claimed invention.

14. Claims 1, 8, 11, 15 and 17 stand rejected under 35 U.S.C. 102(b) as set forth in Paper No. 15, mailed 6-28-01 as being anticipated by Mirzayans et al., Am. J. Hum. Genet., 61:111-19, 1997.

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Mirzayans et al., teach genomic-mismatch scanning using PCR amplified DNA which identifies the human chromosomal region 6p25 containing the locus for anterior segment dysgenesis or Iridogoniodysgenesis which results in juvenile glaucoma, see in particular Abstract and Chromosome 6 Marker Results with GMS. Although the reference fails to disclose a FREAC3 gene or mutation the nucleic acids analyzed at 6p25 meet the limitations of a genetic nucleic acid sequence which may be recognized or named such by the skilled artisan absent factual evidence to the contrary or defining structural limitations of FREAC3. Applicants comments with respect to Watson are noted. However, Watson has not been properly made of record and thus has not been formally considered. Thus, the reference teachings anticipate the claimed invention.

Applicant's argue that Mirzayans is similarly not enabling for the IGDA mutation noted to cause anterior segment dysgenesis because they only isolated the mutation to within 6.9 cM of nucleic acid sequence and that the sequences represents multiple genes. Applicants further argue that no specific mutation is defined and that in contrast the instant invention encodes a polypeptide within 1662 nucleotides which have identified structural and functional domains as well as allelic variants and inactivating mutations. Applicants argue that Watson note that cloning a gene 5cM away from a genetic marker is not trivial.

Applicant's arguments filed 1-11-02 have been fully considered but are not persuasive. In particular it is pointed out that as applicants argue and the examiner agrees (see enablement rejection above), the skill in the art of nucleic acid analysis is high. Mirzayan evidences isolation

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of two separate nucleic acid sequences located at the 6p25 segment, one which possess a mutation that causes anterior segment dysgenesis and one which is normal, and/or not mutated in a way to cause such disease. It is further noted that the breadth of the claims includes any degree of variation for the mutated sequence and that the nucleic acid comparison required to determine the actual sequences of the 6.9 cM segment involves sequencing techniques such as "walking" which are routine in the art, see in particular Kleff et al., EMBO J., 11(2):699-704, 1992 and Shirley et al., Plant Cell 4(3):333-47, 1992, and is capable of defining the specific differences amongst the isolated sequences. In addition, it is noted that the sequence characteristics of the claims are inherently provided as the FREAC3 gene contained within the chromosomal region is deemed to be at least 90% identical to SEQ ID NO:2, absent convincing factual evidence to the contrary. Applicants comments with respect to Watson are noted. However, Watson has not been properly made of record and thus has not been formally considered. It is noted that the Kleff and Shirley references provide support as to the skill in art of chromosomal walking and nucleic acid sequence analysis. Thus, the reference teachings anticipate the claimed invention.

Rejections Necessitated by Amendment

15. Claims 1-5, 8, 11 and 15-17 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. This is a new matter rejection.

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Applicants have amended claim 1 to newly recite, "and said FREAC3 gene encodes a polypeptide that is 90% identical to SEQ ID NO:2." The recitation is noted to be supported at p. 8, lines 14-18 and p. 14, lines 15-23. Yet p. 8, lines 14-18 defines a FREAC3 gene as disclosed and p. 14, lines 15-23 define the term "substantially identical" as disclosed. However, FREAC3 is only disclosed as corresponding to the sequence denoted in Figure 2, i.e., SEQ ID NO:1. There is no apparent support for FREAC3 of 90% sequence identity as disclosed in the specification. Thus, the recitation appears to constitute new matter absent evidentiary support for a FREAC3 of 90% identity to SEQ ID NO:2.

Status of Claims

16. No claims are allowed.

Conclusion

17. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event,

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however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

18. Any inquiry of a general nature or relating to the status of this general application should be directed to the Group receptionist whose telephone number is (703) 308-0196.

Papers relating to this application may be submitted to Technology Center 1600, Group 1640 by facsimile transmission. The faxing of such papers must conform with the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). Should applicant wish to FAX a response, the current FAX number for Group 1600 is (703) 308-4242.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Sharon L. Turner, Ph.D. whose telephone number is (703) 308-0056. The examiner can normally be reached on Monday-Thursday from 8:00 AM to 6:30 PM. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Kunz, can be reached at (703) 308-4623.

Sharon L. Turner, Ph.D.
March 6, 2002

Gary L. Kunz
GARY L. KUNZ
SUPERVISORY PATENT EXAMINER
TECHNOLOGY CENTER 1600